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Decrement in Postural Control During Mild Hypobaric Hypoxia

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The effects of mild hypoxia on the postural control system of 39 naive subjects were examined by measuring the postural sway with a Kistler force platform, at ground level and at one of four simulated altitudes: 1,521 m (5,000 ft), 2,438 m (8,000 ft), 3,048 m (10,000 ft), or 3,658 m (12,000 ft). The total sway increased above the ground level controls for the 1,521 m, 2,438 m, and 3,048 m exposures ($p \leq 0.005$) as did the sway at the lowest measurable frequency ($p \leq 0.002$), but no change in sway was seen in those subjects exposed to 3,658 m as compared to ground level values. Significant interaction between altitude and exposure was observed at $p \leq 0.04$, reflecting the definite effect at the lower altitudes and the lack of an effect at the higher altitude. The multiple comparison test indicated no difference between the responses at 1,524 m, 2,438 m, and 3,048 m. Both arterial oxygen saturation, S_aO_2, and the end-tidal oxygen partial pressure, $P_{et}O_2$, decreased in relation to the test altitudes with a statistically significant interaction between altitude and $P_{et}O_2$ ($p \leq 0.02$), and S_aO_2 ($p \leq 0.005$). There was no significant interaction between altitude and end-tidal carbon dioxide partial pressure ($p = 0.4853$). The postural control mechanisms, as an intergrative functional unit, are very sensitive to acute mild hypoxia. Arguments are advanced to indicate that intervention of compensatory mechanisms at higher altitudes may explain the recovery of postural stability at 3,658 m.

THE MAJORITY of the studies on mild hypoxia in humans have concentrated on various performance indicators to determine if there are modifications to central nervous system (CNS) function (3,7,9). Other research on the physiological responses to mild hypoxia has indicated impairment of night vision at altitudes as low as 2,242 m (7,300 ft) (19). In spite of the importance of the vestibular and postural control systems to pilot performance and flying safety, especially under conditions of reduced visibility where visual inputs to the vestibular system are reduced or distorted, few studies have addressed the effects of mild hypoxia on vestibular end-organ and CNS processing of postural control input and output. Earlier investigations have indicated that neither vestibular end-organ nor hearing functions are particularly sensitive to hypoxia (26), although Vaernes *et al.* (25) have reported complaints of dizziness at altitudes as low as 3,048 m (10,000 ft).

The paucity of information on this aspect of the effects of mild hypoxia results from, in part, the use of clinical techniques designed to detect gross pathological abnormalities of the vestibular system. Such tools are not necessarily sensitive enough to detect the more subtle effects on the postural control mechanisms. The assessment of postural control, as measured by total postural sway, is a sensitive indicator of the direct effects of hyperbaric exposure (1) and alcohol (8,15) on the CNS. It is also capable of showing a decrement in balance control long after subjective and other performance tests have failed to detect any residual CNS action due to anesthetics (17). Postural control can be assessed in a rapid, simple way using a static force platform that provides a quantitative measure of body sway. It is sensitive to any disturbances in information input via the visual, vestibular, and proprioceptive end-organs, higher-order processing and integration in the brain, and motor output that controls the musculoskeletal apparatus (20). All these subsystems have a direct role in the maintenance of postural stability. The changes observed in the various frequency components of sway may indicate which part of the postural control

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mechanism is being affected by a given environmental stress (28).

In this study, a static force platform was used to assess the effects of four different mild hypobaric hypoxia exposures on the postural control capabilities of naive human subjects. The total body sway over all frequencies, and the sway at the lowest measurable frequency with the eyes closed, were evaluated at each simulated altitude.

METHODS

There were 39 naive, male, Canadian Forces (CF) personnel, ranging in age from 18 to 27 years, who volunteered for this study. Each subject provided informed written consent. All candidates had recently completed basic training in the CF and were awaiting permanent posting. None flew on a regular basis or had previous experience in a hypobaric chamber. The experiments were conducted in a hypobaric chamber at normal ambient temperature. Prior to the start of the experiments the subjects were exposed to a simulated altitude of 1,521 m (5,000 ft) and returned to ground level without their knowledge for the control trials. The subjects were then taken to one of the four test altitudes —1,521 m (5,000 ft), 2,438 m (8,000 ft), 3,048 m (10,000 ft) or 3,658 m (12,000 ft), and stayed at altitude for at least 30 min before being tested on the force platform. A subject was exposed to only one of the four test altitudes.

The subjects performed a series of trials, identical to the protocol developed by Black *et al.* (4) for clinical evaluation purposes, by standing in their stocking feet on the force platform with feet together (Romberg position), or heel-to-toe (modified Romberg), while doing the Jendrassik maneuver (the fingers of the hands are hooked together and a light isometric pull is exerted). This forces the body to behave as an inverted pendulum with minimal movement of the upper portions. Four 15-s trials were performed with the eyes closed over the course of 3–4 min.

A quartz multicomponent force measuring platform (Model 9261A, Kistler Instruments, Switzerland) was used to monitor the displacement of the centre-of-gravity of each subject. The signals from the force platform were fed to a Compact System Controller (CSC) of a DEC PDP 11/04 minicomputer. The data were collected and analysed using the software package developed by Black *et al.* (5) with the data collection and graphics routines modified in-house, in order to interface with the laboratory graphics terminal (Hewlett-Packard HP2647A) and printer-plotter (Hewlett-Packard HP2631G). Two sway parameters were obtained from each force platform run. The total power of the radial sway vector (PS_{tot}) was calculated by integrating the area under the power spectrum of the radial sway (Fig. 1). The radial sway is the vector connecting the centre-of-force application to the average centre-of-force application for a given force platform trial.

A typical display of the centre-of-force application over a 15-s force platform trial is given in Fig. 2. The second sway parameter calculated was the power of the sway at the lowest measurable frequency of 0dB. The final values were derived from the average of the four trials.

The end-tidal partial pressures of oxygen, $P_{et}O_2$, carbon dioxide, $P_{et}CO_2$, and the arterial oxyhemoglobin saturation, S_aO_2, were determined immediately prior to each series of balance tests. $P_{et}O_2$ and $P_{et}CO_2$ were measured with a mass spectrometer (Medspect II, Searle) and S_aO_2 with an ear oximeter (Hewlett-Packard 47201A).

All data were transferred to a VAX 750 computer. A two-way analysis of variance (ANOVA) for repeated measures was performed on the data using one grouping factor (the altitude exposure) and one within factor (the pre-altitude, altitude, or post-altitude exposure). The calculations were made with the BMDP-P2V program. The linear contrast comparison (22) was chosen as the test statistic with ω as one of the following measured variables: PS_{tot}, 0dB, $P_{et}O_2$, $P_{et}CO_2$, or S_aO_2. The combined values of the pre-altitude and post-altitude conditions were compared to the altitude exposures. Combining the pre- and post-altitude exposures should correct for any learning effect with repeated platform testing, which has been observed by both Holliday and Fernie (16)

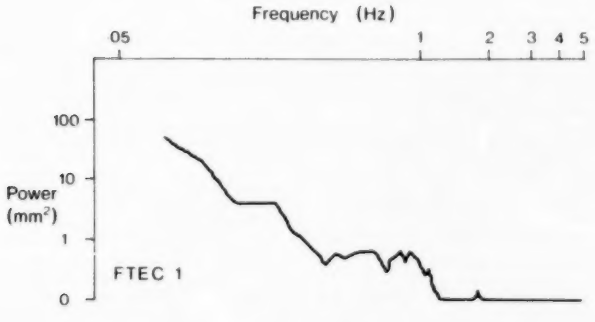

Fig. 1. An example of a plot of the power of the total sway (mm^2) versus the sway frequency (Hz). The power of the sway at the lowest measureable frequency is the 0dB parameter.

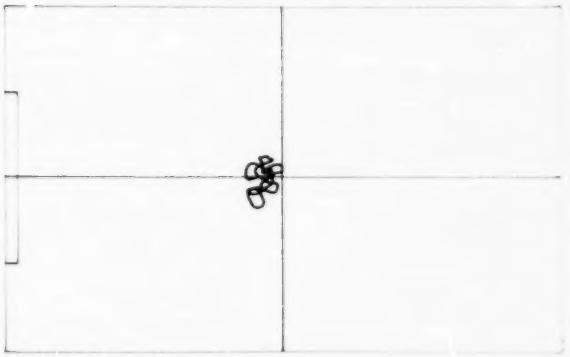

Fig. 2. Hard copy of a complete 15-s force platform-trial. The subject's centre-of-force trajectory is traced to scale on the schematic of the force platform.

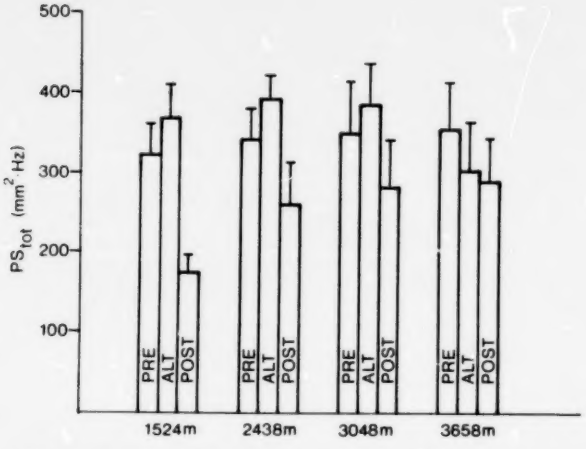

Fig. 3. A plot of the total power of the radial sway vector (PS_{tot}) for the pre-altitude (PRE), altitude (ALT), and post-altitude (POST) conditions. The subjects were exposed to one of the 4 altitudes: 1,521 m (5,000 ft), 2,438 m (8,000 ft), 3,048 m (10,000 ft), or 3,658 m (12,000 ft).

and Black (5). The null and alternate hypotheses for each variable are H_0: $\omega_{pre} + \omega_{post} - 2 \cdot \omega_{altitude} = \epsilon = 0$; and H_1: $\omega_{pre} + \omega_{post} - 2 \cdot \omega_{altitude} = \epsilon \neq 0$. Multiple comparisons of the ω's across the altitudes were done using the Student-Newman-Keuls test (22). The difference was considered significant when the probability exceeded the 95% level (i.e., $p<0.05$).

RESULTS

The total sway increased above the ground level controls for the 1,521 m, 2,438 m, and 3,048 m exposures, but no change in sway above ground level values was seen in those subjects exposed to 3,658 m altitude (Fig. 3, Table I).

The ANOVA results are given in Table II. The null hypothesis—no effect due to altitude—was rejected at the level of $p \leq 0.005$. Significant interaction, reflecting the definite effect at the lower altitudes and the lack of an effect at the higher altitude, was observed ($p \leq 0.04$).

A multiple comparison test indicated no difference between the responses at 1,524 m, 2,438 m, and 3,048 m: $PS_{tot\ 1524\ m} = PS_{tot\ 2438\ m} = PS_{tot\ 3048\ m} > PS_{tot\ 3658\ m}$.

The null hypothesis for 0dB was rejected ($p \leq 0.002$). Significant interaction was seen between altitude and 0dB ($p \leq 0.03$) (Table II). As for PS_{tot}, the low frequency sway increased at altitudes of 1,524 m, 2,438 m and 3,048 m, but there was no observable effect at 3,658 m ($p<0.10$). The multiple comparisons test indicated that there were no detectable differences between the three lower altitudes ($p>0.10$).

The values for S_aO_2, $P_{et}O_2$, and $P_{et}CO_2$ were all significantly affected at altitude (Table 1). Both S_aO_2 and $P_{et}O_2$ decreased in relation to the test altitudes with a statistically significant interaction between altitude and $P_{et}O_2$ ($p \leq 0.002$) and S_aO_2 ($p \leq 0.005$). There was no significant interaction between altitude and $P_{et}CO_2$ ($p>0.10$).

TABLE I. VALUES OF THE SWAY AND PHYSIOLOGICAL PARAMETERS

	Altitude											
	1524 m (5,000 ft) n = 9			2438 m (8,000 ft) n = 11			3048 m (10,000 ft) n = 8			3658 m (12,000 ft) n = 11		
	Pre	Alt	Post	Pre	Alt	Post	Pre	Alt	Post	Pre	Alt	Post
PS_{tot} $mm^2 \cdot Hz$ ± S.E.	320 ± 40	367* ± 50	175 ± 30	341 ± 39	391* ± 30	258 ± 55	349 ± 66	385* ± 50	281 ± 62	352 ± 64	301 ± 64	289 ± 55
Zero 0 dB mm^2 ± S.E.	45 ± 5	48* ± 6	23 ± 5	43 ± 6	56* ± 7	30 ± 7	43 ± 8	51* ± 7	35 ± 11	48 ± 12	38 ± 9	40 ± 9
$P_{et}O_2$ mm Hg ± S.E.	118.7 ± 1.5	92.4* ± 2.5	112.4 ± 3.2	122.6 ±3.3	85.7* ± 5.3	123.3 ± 4.8	123.0 ± 2.1	71.6* ± 4.7	120.8 ± 2.1	120.2 ± 3.8	70.7* ± 7.3	118.5 ± 3.8
$P_{et}CO_2$ mm Hg ± S.E.	34.1 ± 2.2	33.4* ± 2.8	34.6 ± 2.5	38.2 ± 0.8	35.3* ± 1.1	36.0 ± 1.7	37.0 ± 1.2	32.8* ± 2.1	36.1 ± 1.3	38.4 ± 0.8	35.1* ± 1.5	36.9 ± 1.1
S_aO_2 ± S.E.	96.2 ± 0.3	94.7* ± 0.4	96.3 ± 0.3	96.2 ± 0.4	91.5* ± 0.5	96.3 ± 0.3	96.8 ± 0.3	90.3* ± 0.8	96.9 ± 0.4	97.0 ± 0.3	87.3* ± 1.6	96.1 ± 1.1

(PS_{tot}), total power of the radial sway, (0 dB) the power of the sway at the smallest measurable frequency, ($P_{et}O_2$) the end-tidal oxygen partial pressure, ($P_{et}CO_2$), the end-tidal carbon dioxide partial pressure (S_aO_2) the arterial oxyhemoglobin concentration for pre-altitude, altitude, and post-altitude conditions. n = number of subjects exposed to a given altitude. All values are as the mean ± standard error of the mean.

* Significantly different from combined pre- and post-altitude exposure values at $p < 0.05$.

TABLE II. TWO-WAY ANALYSIS OF VARIANCE.

Source	SSq	DF	MSq	F	Tail Probability
Exposure	2610	1	2610	11.66	0.0016
Exposure × Altitude	2357	3	786	3.51	0.0249
Exposure (PS_{tot})	109510	1	109510	13.81	0.0007
Exposure × Altitude	2258	3	752	3.84	0.0184
Exposure (P_{etO_2})	34867	1	34867	177.70	0.0001
Exposure × Altitude	2258	3	752	3.84	0.0184
Exposure (P_{etCO_2})	143	1	143	12.56	0.0011
Exposure × Altitude	28	3	9	0.83	0.4853
Exposure (S_aO_2)	847	1	847	85.05	0.0001
Exposure × Altitude	211	3	70	7.05	0.0007

Two-way ANOVA for repeated measures of the power of the radial sway at the minimum frequency (0dB), the total power of the radial sway vector (PS_{tot}), the end-tidal oxygen partial pressure (P_{etO_2}), the end-tidal carbon dioxide partial pressure (P_{etCO_2}), and the arterial oxyhemoglobin saturation (S_aO_2) using one grouping factor (altitude exposures) and one within factor (pre, altitude, and post).

DISCUSSION

Acute mild hypoxia induced by exposures to simulated altitude produced a significant increase in body sway. Even an altitude as low as 1,524 m resulted in a significant impairment in postural stability. These results substantiate the anecdotal observations of Vaernes *et al.* (25) on the feelings of dizziness expressed by subjects during long exposures to relatively low altitudes. They also agree with other studies (24,28) that reported an increase in the low-frequency components of body sway during exposures to an altitude of 3,000 m (9,750 ft).

The decrease in arterial oxygen partial pressure P_aO_2 due to altitude, may affect neural processing at the vestibular end-organ, the vestibular nuclei of the brain stem, the neuromuscular junctions of the skeletal system, the vestibular cortical processing segments of the brain, or the proprioceptive input. Yamazaki *et al.* (28) have argued that the lower frequency components of the body sway represent the constituents of the body's sway under vestibular control. If this is true, then the variations in the amplitude of the sway, as measured by changes in the 0dB parameter, indicate that the vestibular component of the postural control is one of the affected subsystems. The force platform does not allow one to determine if this effect is localized at the vestibular end-organ, in the brain stem, and/or in the cerebral cortex. Earlier studies, have shown that the auditory portion of the inner ear is relatively insensitive to hypoxia (23), whereas the auditory cortex is more readily disturbed by moderate hypoxic exposure (11). The same cortical sensitivity for the vestibular system might be expected since the higher levels of the CNS are, in general, more sensitive to hypoxia (10).

Even under conditions of moderate-to-severe hypoxia, there are no detectable changes in O_2 consumption or energy substrate concentrations (i.e. ATP) in the brain (2,12). On the other hand, mild hypoxia has a substantial impact on other biochemical processes in brain cells including neurotransmitter synthesis. It is difficult to accept that an arterial oxygen partial pressure of 75 mm Hg, corresponding to an altitude of 1,524 m, could affect the biochemical processes in any portion of the brain. However, the rate of O_2 consumption in the cerebral tissue is sufficient to reduce the brain tissue oxygen tension (P_{bO_2}) to as low as 19 mm Hg (18). Certain cerebral cells in the more distal portions of the Krogh diffusion cylinder surrounding the brain capillaries may be close to the anoxic state, even under normal conditions. Therefore, small decreases in inspired O_2 partial pressures (P_IO_2) in the absence of any compensatory changes in cerebral blood flow and/or cell metabolism, could induce a subtle shift to anaerobic metabolism and/or changes in the synthesis and degradation of nonenergy-related cell metabolites. Gibson and Duffy (14) examined the effects of mild hypoxia by exposing rats to 15% and 10% O_2 gas mixtures balanced with nitrogen. They found a 45% increase in glucose utilization and a corresponding rise in lactate concentration in the cerebral tissue of rats exposed to the 15% O_2. This marked effect on glycolysis was accompanied by a decrease in the synthesis of acetylcholine. Synthesis from glucose and choline was reduced by 46% and 38%, respectively. Gibson *et al.* (13) observed similar reductions in acetylcholine metabolism and in the synthesis of a number of amine neurotransmitters in mice. Modifications of this sort in the human subjects involved in this study could account for the deterioration in postural stability, since it has been established that only subtle modifications of neurotransmission in the brain can have a significant impact in vestibular and postural functions (8). Such changes in brain metabolism could explain the other neurophysiological effects caused by hypoxia, such as the decreased dark adaptation capability at altitudes as low as 2,242 m (7,300 ft) (19), and the decreased cortical processing of the auditory stimulus at low altitude (11).

No significant alteration in postural stability was

found at an altitude of 3,658 m. This lack of effect is possibly the result of compensatory mechanisms that become effective at this moderate degree of hypoxia. For example, the cerebral blood flow will begin to increase substantially at this level of hypoxia (6,27), though it may require several minutes for the maximum rise to occur. Meanwhile, the lactate accumulation seen by Gibson and Duffy (14) would take place. The increased cerebral blood flow would compensate for the fall in $P_{a\bar{v}}O_2$, and $P_{b\bar{v}}O_2$ would then be maintained closer to the ground level control values. Thus, brain chemistry at an altitude of 3,658 m could be similar to ground level controls, whereas the less severe hypoxic exposures would result in greater disturbances in brain chemistry, since the hypoxia is below the thresholds for compensatory mechanisms. This hypothesis is supported by the findings of Gibson and Duffy (14) that a 10% O_2-90% N_2 gas mixture did not increase glucose utilization in rat brain cortical tissue above that seen in control conditions with 30% O_2-70% N_2 whereas the less severe exposure of 15% O_2-85% N_2 did lead to an elevated glucose utilization.

The decrease in postural stability at the very mild levels of hypoxia suggests that more attention should be paid to the effects of altitude exposure on the vestibular apparatus of pilots. The ability of pilots to maintain proper aircraft orientation may become impaired, in those conditions where visual cues are distorted or reduced, if they are not breathing gas of an O_2 concentration sufficient to prevent mild hypoxia.

Further investigations are required to identify the components of the postural control mechanisms that are the most affected by mild hypoxia. The interaction between mild hypoxia and drugs (social or prescription) should also be investigated. Since the response to mild hypoxia is greatly dependent on the glucose substrate levels in the cerebral blood supply, a hypoglycemic condition would have an obvious interaction with any hypoxic stimulus to increased glucose utilization.

The analysis of the effects of mild hypoxia, drugs, or carbon monoxide, may be more relevant if both performance and physiological parameters are measured when the brain functions are highly stimulated and both cerebral glucose and O_2 utilization are increased.

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